Objectives: To analysis the correlation of coronary artery stenosis and cerebral vascular stenosis, to explore the clinical significance in early diagnosis and treatment for patients with cardiovascular disease.
Methods: From 2010 to 2012, The 146 hospitalized patients with cardiovascular patients or suspected cardiovascular disease come for treaments, excluded age larger than 80 years, severe heart liver and kidney dysfunction, severe bleeding tendency or bleeding disorders, allergy to contrast agents or in patients with contraindications. investigating the patient's age, gender, smoking, alcohol consumption, history of diabetes, history of hypertension, a history of high cholesterol and other risk factors, and then the elective coronary angiography (CAG) and digital subtraction angiography (DSA) of the whole brain cardiovascular and cerebrovascular lesions were applied for diagnosis of the patients.
Results: (1) The occurrence of coronary artery stenosis were relative to the occurrence of cerebral vascular stenosis, the Kappar coefficient was 0.53 (0.23 to 0.84), and cerebral vascular stenosis incidence gradually increased with increasing coronary stenosis count. (2) There was a positive correlation between coronary artery stenosis count and the number of cerebral vascular stenosis, the Spearman coefficient was r=0.62. (3) The difference of cerebral vascular stenosis between the single, double and three coronary artery stenosis patients was not statistically significant. (4) The coronary artery stenosis in patients with cerebrovascular stenosis risk OR value (95% CI) was 16.5 (2.92-93.20); adjusted for sex, age, the OR value (95% CI) changed to 18.45 (1.65-205.97) , and the model predictive ability (AUC=0.929). As coronary stenosis count increased the cerebral vascular stenosis increased risk OR (95% CI) was 10.44 (1.32-82.85).
Conclusions: The occurrence of coronary stenosis may occur cerebral blood narrow simultaneously, So early cerebrovascular inspection, prevention, targeted therapy for patients with coronary artery stenosis were necessary.

GW25-e2238

The mechanism of leukoaraiosis: multiple micro-leuko-infarction might play a role

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Objectives: To investigate the association of micro-leuko-infarction and leukoaraiosis. Leukoaraiosis is a major cause of vascular dementia and disability, but the mechanism and etiology is unclear.
Methods: Collect the data of the patients without overt neurological symptoms and signs and happened to have brain MRI examination and finding new dotted lesions in white matter on DWI-sequenc. Eighteen patients with vertigo or dizziness, memory impairment etc, but without overt neurological symptoms and signs, who happened to find new dotted lesions in white matter on DWI sequence of MRI were concluded. MRI assessment included the severity of white matter changes and the numbers of lacunes. The atherosclerosis of carotid and cerebral vascular were examined with MRA, ultrasound, or DSA.
Results: MRI DWI sequences showed bright dotted lesions, located in the surroundings of lateral ventricle, mainly in corona radiate. T2/FLAIR sequence: 2 patients showed no cloudy white matter changes along the antecornu and postcornu and body of lateral ventricle; 16 patients showed varied degrees of luekoaraiosis, 5 of them had multiple punctuate dotted lesions of subcortex or deep white matter. The patients had intima-media thickness, or single or multiple carotid artery atherosclerotic plaques, different degree of vascular atherosclerosis. Patients with severe atherosclerosis were often accompanied with severe white matter lesions.
Conclusions: Our results reinforce the close association between LA and micro-leuko-infarction.

Pulmonary Circulation

GW25-e2277

Remodeling of right heart in rats pulmonary arterial hypertension induced by monocrotaline

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Objectives: Pulmonary arterial hypertension (PAH) is a severe disease that could cause dysfunction of right heart finally. The right heart is the target organ of PAH injury. So that the remodeling of right heart plays an important role in the progression of PAH. Monocrotaline (MCT) could induce experimental PAH. While the changes of right heart of rats PAH induced by MCT were rarely reported including cellar hypertrophy and proliferation. We studied the remodeling of right heart in rats PAH induced by MCT.
Methods: The rats PAH was induced by a single subcutaneous injection of MCT 50 mg/kg, control group was injected with normal saline. MCT-PAH rats were randomly divided into three groups according to the treatment of MCT: 2 weeks, 3 weeks and 4 weeks (MCT-2w, MCT-3w and MCT-4w). The hemodynamics and right ventricular hypertrophy were detected in each group. The weights of the free wall of the right ventricle(RV) and the left ventricle plus septum(LV+S) were measured, and the ratio of RV/(LV+S) was calculated as the RV hypertrophy index(RVHI).The expression levels of proliferating cell nuclear antigen(PCNA) were detected in right ventricular tissue.
Results: After MCT injection for three weeks, mean pulmonary arterial pressure, right ventricular systolic pressure were significantly increased. RVHI in MCT -3w and MCT -4w was significantly increased compared to control (both $P<0.05$). Pathology results showed the vascular intimal hyperplasia of pulmonary vasculature in MCT groups. In control, the nuclei of cardiomyocytes of right heart were clear and in alignment with consistent muscular fiber direction. While in MCT -3w and MCT -4w, cardiomyocytes of right heart were enlarged with disorder arrangement, sarcoplasm dissolved in some myocytes with increased interstitial. The PCNA-positive cells in right ventricle tissue of MCT groups were significantly increased compared to control ($P<0.05$, respectively).
Conclusions: There is hypertrophy of cardiomyocytes and interstitial increasing in right heart of MCT induced PAH rats. It suggests the remodeling of right heart in the experimental PAH.

GW25-e2325

Mfn2 down-regulation promotes PASMCs proliferation in hypoxic pulmonary hypertension rats via PI3K/Akt pathway and mitochondria apoptosis pathway

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Objectives: Pulmonary arterial smooth muscle cells (PASMCs) excessive proliferation is one of the most important pathological features of hypoxic pulmonary vascular remodeling (HPVR) in hypoxia pulmonary hypertension (HPH). Many researches have found Mitofusin 2 (Mfn2) played an important role in the pathogenesis of Hypertension, but whether it palyed the same role in the HPH is unkown yet. We investigated the expression of Mitofusin 2 (Mfn2) and its roles in the balance of PASMCs proliferation and apoptosis via PI3K/Akt signal pathway, and mitochondria apoptosis pathway under hypoxic condition.
Methods: In vivo, after the rats were exposed to hypoxia (10±0.5)% and normoxia (20%) for 4 weeks respectively, the expression of Mfn2 and proliferating cell nuclear antigen(PCNA) in the rats lung tissues were tested by reverse transcription-PCR (RT-PCR) and Western Blot. In vitro, the primary cultured PASMCs from rats pulmonary arterioles were being cultured under normoxia (2.5% O_2/5%CO_2/ balance N_2)and hypoxia incubator(21% O_2/ 5%CO_2/ balance N_2). The proliferation and cell cycle of PASMCs were tested by the MTT assay and flow cytometry assay. Furthermore, we over-expressed Mfn2 by the plasmid which was carried pEGFPMfn2 cDNA and transfected by Lipofectamine^{TM}2000 and/or treated the PASMCs with LY294002 (PI3K inhibitor) before cultured in hypoxia for 24h. Then the expression of Mfn2, p-Akt (phosphorylation type of Akt), mitochondrial cytochrome C in cytoplasm and mitochondria, cleaved caspase 9 were tested by Western Blot with β-actin as internal protein.
Results: The expression of Mfn2 mRNA and protein were reduced and the PCNA protein was up-regulated in the HPH rat lung tissue. Under hypoxic conditions, the results of MTT assay and flow cytometry assay showed the number of PASMCs increased and more cells entered the cell cycle of S+G2/M phase in hypoxia compared with normoxia. Furthermore, Mfn2 down-regulation increased the expression of p-Akt with the PI3K/Akt signaling pathway activating which resulted the expression of PCNA increased, more cells entered into the cell cycle of S+G2/M, the ratio of cytochrome C expression in cytoplasm to mitochondrial and the expression of cleaved caspase 9 decreased. However, when Mfn2 were over-expressed or/and the PASMCs treated with LY294002 (PI3K inhibitor) under hypoxic condition, all of the effects above were reversed.
Conclusions: Mfn2 down-regulated in the HPH rat lung tissue and in PASMCs under hypoxic condition and induced the PI3K/Akt pathway activation, more cells entered the S+G2/M phase of cell cycle and inhibited the mitochondrial apoptosis pathway.

GW25-e4451

Effect of nicorandil on the chronic air embolism-induced pulmonary hypertension and pulmonary vascular remodeling in rabbits

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Objectives: To investigate the effect of nicorandil on the chronic air embolism-induced pulmonary hypertension and pulmonary vascular remodeling in rabbits.
Methods: A total of 36 rabbits were randomly divided into control group, air embolic group, and air embolic + nicorandil treated group with 12 rabbits 0f each group. In the latter two air embolic groups of rabbits filtered air were continuously infused through ear vein about 1ml(0.05ml/min/kg), which were recevied continuous air embolization